Atypical Herpetic Corneal Endotheliitis: A Case Report

Atipik Herpetik Korneal Endotelit: Olgu Sunumu

Herpetic Endotheliitis

Aylin Karalezli¹, Ali Kucukoduk², Banu Bozkurt³ ¹Department of Ophthalmology, Mugla Sitki Kocman University, School of Medicine, Mugla, ²Department of Ophthalmology, Baskent University, School of Medicine, Ankara, ³Department of Ophthalmology, Selcuk University Faculty of Medicine, Konya, Turkey



Özet	Abstract
Korneal endotelit, sıklıkla viral orijinli olan ve primer olarak kornea endotelinin if-	Corneal endotheliitis; frequently caused by viruses, is a disorder in which corneal
lamasyonunun gözlendiği bir hastalıktır. Bu olgu sunumunda ön kamara reaksiyo-	endothelium is the primary site of the inflammation. We discuss a case of atypi-
nunun gözlenmediği sadece tek taraflı yaygın korneal ödem ile giden atipik herpe-	cal herpetic endotheliitis presented with unilateral diffuse corneal edema with no
tik korneal endotelitli bir olguyu sunuyoruz.	signs of anterior chamber reaction.
Anahtar Kelimeler	Keywords
Korneal Ödem; Endotelitis; HSV	Corneal Edema; Endotheliitis; HSV
nunun gözlenmediği sadece tek taraflı yaygın korneal ödem ile giden atipik herpe- tik korneal endotelitli bir olguyu sunuyoruz. Anahtar Kelimeler Korneal Ödem; Endotelitis; HSV	cal herpetic endotheliitis presented with unilateral diffuse corneal edema wit signs of anterior chamber reaction. Keywords Corneal Edema; Endotheliitis; HSV

DOI: 10.4328/JCAM.4089Received: 13.11.2015Accepted: 14.12.2015Printed: 01.01.2016J Clin Anal Med 2016;7(1): 141-3Corresponding Author: Aylin Karalezli, Department of Ophthalmology, Sitki Kocman University School of Medicine, 48000, Mentese, Mugla, Türkiye.T.: +90 2522115211 E-Mail: akaralezli@yahoo.com

Introduction

Corneal endotheliitis can be defined as a broad range disorder in which corneal endothelium is the primary site of the inflammation and is characterized by corneal edema, keratic precipitates (KPs), and mild anterior chamber reaction [1]. The disease is also characterized by the lack of inflammatory changes in the corneal stroma and intraocular pressure (IOP) elevation; therefore, secondary impairment of corneal endothelium due to stromal or interstitial keratitis can be ruled out [2]. Herpes simplex virus (HSV), cytomegalovirus (CMV), varicella zoster virus (VZV), rubella virus, and mumps infection are considered to be potentially causative viruses of corneal endotheliitis [3]. We present a case of atypical HSV endotheliitis that presented with unilateral diffuse corneal edema with no other signs of anterior chamber reaction such as IOP elevation, keratic precipitates, or anterior chamber reaction.

Case Report

A 24-year old female patient was admitted to our clinic with complaints of recurrent blurred vision in the left eye lasting for one week within the previous 2-month period. The patient had no history of trauma, ocular surgery or systemic diseases. Best corrected visual acuity was 20/20 in the right eye and 20/200 in the left eye. On slit-lamp examination, a diffuse corneal edema with descemet folds was observed in the left eye with no evidence of anterior chamber inflammation and keratic precipitates (Figure 1). Anterior segment examination of the right



Fig. 1 Corneal edema with no anterior chamber inflammation

eye was normal. Intraocular pressures were 16 mmHg in both eyes. Posterior segment examination was normal in the right eye, but because of corneal edema, could not be done in the left eye. Central corneal thicknesses were 553 µm and 777 µm in the right and left eyes, respectively. Endothelial cell density was 2771 cells/mm2 in the right eye and 1454 cells/mm2 in the left eye with high polymegathism by specular microscopy (Figure 2). Corneal hypoesthesia was remarkable in the left eye compared to the right eye.

Because of recurrences in the same eye and hypoesthesia, a diagnosis of HSV corneal endotheliitis was suspected and treatment with topical 3% acyclovir ointment 5 times a day, topical prednisolone acetate 1% 5 times a day and oral acyclovir 5x400 mg was started. After 2 weeks of treatment, the visual acuity of



Fig 2. Decreased endothelial cell density with high polymegathism

the left eye was 20/20, corneal edema was resolved, descemet folds disappeared, and central corneal thickness lowered to 542 μm (Figure 3). Three weeks later, topical acyclovir and prednisolone acetate were ceased and 800 mg oral acyclovir was started to prevent recurrences. The patient has had no recurrences of corneal edema for the last 6 months.



Fig 3. 2 weeks after treatment, corneal edema resolved

Discussion

Corneal transparency is maintained by many factors, including the ultrastructural anatomy and the physiology of the cornea and its cellular and extracellular components. Corneal trauma, surgeries, diseases, and infections may lead to persistent corneal opacities. Corneal endothelium plays an important role in maintaining corneal transparency and its abnormality or dysfunction results in corneal edema.

Ocular trauma, surgeries, Fuchs endothelial dystrophy, iridocorneal endothelial syndromes (ICE), stromal keratitis, and endotheliitis should be considered in the differential diagnosis of corneal edema [2,4-8]. In our case, the absence of any ocular trauma or surgery, no signs of cornea guttae, and a normal specular microscopic examination in the fellow eye, and the lack of iris findings and typical specular findings for ICE syndrome in the left eye, led us to the diagnosis of endotheliitis of viral origin. Corneal endotheliitis is characterized by corneal edema, KPs, and mild anterior chamber reaction with no signs of inflammatory changes in the corneal stroma [2]. HSV, CMV, VZV, rubella virus, and mumps are considered to be causative viruses of endotheliitis [3]. Clinical diagnosis can be supported by the detection of viral nucleic acid in an aqueous sample by polymerase chain reaction (PCR) or intraocular antibody detection, and Goldmann-Witmer coefficient (GWc) calculation. Thus, we recommended an anterior chamber tap, but the patient rejected the procedure. Atypically, the patient had no signs of anterior chamber inflammation such as anterior chamber reaction, keratic precipitates, and IOP elevation.

Bass et al. described 3 patients of atypical herpetic disease presented with unilateral posterior stromal lesions with diffuse stromal edema and the lack of epithelial involvement and posterior stromal opacification [9]. They stated that young patients presenting with unilateral posterior stromal opacification and stromal edema in the absence of epithelial involvement are likely to have endotheliitis of herpetic origin. Chiang et al. reported a patient with bullous keratopathy, without KP, anterior chamber reaction, or IOP elevation caused by CMV endotheliitis [10]. In the study, PCR was used to detect viral DNA in aqueous humor samples. Similarly, Papaioannou et al. described a patient with atypical clinical presentation of viral endotheliitis and proved HSV endotheliitis by positive GWc [11].

In the treatment of corneal endotheliitis, systemic and topical antiviral treatment with topical corticosteroids is a rational strategy [12]. Topical corticosteroids should be used cautiously and preferably with antivirus cover [3]. Long-term treatment with acyclovir helps prevent recurrences of ocular HSV disease and orofacial HSV infections in patients with a history of ocular HSV disease [12]. In our case, the lack of anterior chamber reaction directed us to start the initial therapy with topical acyclovir and steroids, and we continued the therapy with 800 mg oral acyclovir to prevent recurrences.

In summary, in young patients with recurrent unilateral corneal edema with no history of ocular trauma and surgery, herpetic disease should be considered.

Competing interests

The authors declare that they have no competing interests.

References

1. Khodadoust AA, Attarzadeh A. Presumed autoimmune corneal endotheliopathy. Am J Ophthalmol 1982;93:718-22.

- 2. Suzuki T, Ohashi Y. Corneal endotheliitis. Semin Ophthalmol 2008;23:235-40.
- 3. Jap A, Chee SP. Viral anterior uveitis. Curr Opin Ophthalmol 2011;22:483-8.
- 4. Agrawal R, Shah M, Mireskandari K, Yong GK. Controversies in ocular trauma

classification and management: review. Int Ophthalmol 2013;33:435-45. 5. Shoss BL, Tsai LM. Postoperative care in cataract surgery. Curr Opin Ophthalmol 2013;24:66-73.

6. Adamis AP, Filatov V, Tripathi BJ, Tripathi RC. Fuchs' endothelial dystrophy of the cornea. Surv Ophthalmol 1993;38:149-68.

7. Shields MB. Progressive essential iris atrophy, Chandler's syndrome, and the iris nevus (Cogan-Reese) syndrome: a spectrum of disease. Surv Ophthalmol 1979;24:3-20.

8. Holland EJ, Schwartz GS. Classification of herpes simplex virus keratitis. Cornea 1999;18:144-54.

9. Bass SJ, Sherman J, Panariello G, Richter SJ. Atypical herpetic endotheliitis. J Am Optom Assoc 1995;66:577-80.

10. Chiang CC, Lin TH, Tien PT, Tsai YY. Atypical presentation of cytomegalovirus endotheliitis: a case report. Ocul Immunol Inflamm 2011;19:69-71.

11. Papaioannou L, Tsolkas G, Theodossiadis P, Papathanassiou M. An atypical case of herpes simplex virus endotheliitis presented as bullous keratopathy. Ocul Immunol Inflamm 2013;21:475-7.

12. Acyclovir for the prevention of recurrent herpes simplex virus eye disease. Herpetic Eye Disease Study Group. N Engl J Med 1998;339:300-6.

How to cite this article:

Karalezli A, Kucukoduk A, Bozkurt B. Atypical Herpetic Corneal Endotheliitis: A Case Report. J Clin Anal Med 2016;7(1): 141-3.